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United States Navy  
MEDICAL NEWS LETTER

Vol. 37

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No. 11

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Drugs on the Market

Editorial, New Engl J Med 264: 621-622, March 23, 1961.

According to his usual custom—now practically a tradition—Paul de Haen, consultant to the pharmaceutical and allied industries, has issued his annual summary of developments in the drug industry during the past year. The total new products marketed by 109 firms in 1960, numbering 311, were studied; the 1959 count was 315 products from 106 firms, the record having been established in 1955 when the 403 offerings laid on the altar of Panacea by 124 companies were subjected to his analysis. The new single chemicals introduced in 1960 numbered 45, the record having been established in 1950 with 63. The statistical picture has not materially changed, therefore, since 1951 when 86 firms that offered 321 new products, of which 35 were new single chemicals, came under his scrutiny.

A rapid scanning of the field shows that the nonmercurial diuretics are still being elaborated, some of them in combination with new or old tranquilizers, double-barreled-shotgun and even Gatling-gun therapy being still an irresistible factor in the search for health. In addition to the products designed for wringer therapy, new hypotensive agents continue to be introduced, as well as new anti-convulsants for the nation's million and a half epileptics. New discoveries of note in the antibiotic line have been based on the need for an effective subduer of strains of staphylococci that are resistant to the older penicillins.

Mindful that the gastrointestinal tract of navel-centered man is inclined either to turbulence or inactivity, the inspired manufacturers have produced 36 substances, according to Mr. de Haen, intended to alkalize it, reduce its speed of action, reactivate it, aid it with new enzymes, add bile to its content, release its excessive gas and anesthetize it. New cough medicines have been introduced, their introducers mindful, or unmindful, of the old reminder that "it isn't the cough that carries you off but the coffin they carry you off in."

Proprietary names continue to be as fantastic as are many of the generic appellations, except for there being mercifully only one of the latter per drug.

Mr. de Haen introduces some pertinent philosophy regarding the changing pharmaceutical needs of a population, the age composition of which has already changed. A reduction in infant mortality has been the important factor in the quadrupling of the population over 65 years of age, the life expectancy of which is not much greater than it used to be. Nevertheless, the needs of this important age group must be met during a period when its earning power is greatly abridged.

"What is required today," Mr. de Haen concludes, "is not so much innovation in industry and medical practices, but a change in attitude, with the realization that the patient's needs must be served first. That is the challenge of the sixties."

\* \* \* \* \*

Pyelonephritis and the Urologist

Myron H. Nourse, Department of Genito-Urinary Surgery, Indiana University School of Medicine, Indianapolis, Ind. J Urol 85:211-213, March 1961.

Interest concerning pyelonephritis is clearly demonstrated by the extensive publications on the subject. It seems to be out of all proportion, however, to the knowledge gained. In fact, there is much that is misleading and bears a "second look."

We (urologists) turn to the internist regularly to help us evaluate and treat the cardiac, the hypertensive, or the diabetic patient who needs a prostatectomy because we value and appreciate his knowledge and because thus the patient receives better medical care. The urologist also depends upon laboratory workers for accurate interpretation of microscopic slides. By the same token, the internist and the pathologist turn to us for an opinion or treatment when confronted with a urologic problem. Each respects the other for his training, his knowledge, and his experience; therefore, it is important that we work together harmoniously for benefit of the sick.

Presuming then that each contributes a part, it seems reasonable to expect the urologist to evaluate data regarding urinary infection and pyelonephritis because most of the reports are by those of nonurologic groups and reflect upon the judicious use of the "tools of the trade."

For the benefit of some, it is vitally important for them to know that, except within the urethral meatus in both sexes, normally the urinary tract is sterile. Davis states: "If persistent infection occurs in the urinary tract, it will usually mean urinary stasis due to obstruction to the normal urinary flow. All changes produced are reversible in the early stages and irreversible in the late stages. Therefore, it is the duty of the physician to learn to detect the obstruction in its early stages, and relieve it before irreparable damage is done."

O'Connor has reported that in his experience more than 75% of patients with recurrent or persistent urinary tract infections have obstructive lesions, neoplasms, or calculi.

These comments represent fundamental urologic teachings of yesterday and today. They are every bit as sound now as before; I question that this is appreciated by the majority of those who are not in the practice of urology.

Talbot concluded that development of obstructive lesions in the upper urinary tract leads to significant renal infection. This obstruction may be from many causes. Kass found that about 6 to 8% of 2000 pregnant women making their first prenatal visit had asymptomatic bacteriuria and that in more than 40% of these pyelonephritis developed if the patients were untreated. None developed pyelonephritis if appropriate chemotherapy or antibiotics were used.

Persistence of urinary infection is presumptive evidence of obstruction or primary urinary tract disease and this will only be determined after a careful, competent, and adequate urologic survey. This will by necessity require urethral instrumentation. Confidence should be given to those who are properly



trained to use these instruments. Boyce aptly states that "pyelonephritis has become as nonspecific a term as Bright's disease. Is it too much to ask that physicians apply current knowledge rationally and diligently to prevention and therapy of urinary infections?"

O'Connor urges that the need for careful complete diagnostic survey of the entire urinary tract has not been changed by the efficiency of modern antibiotics. He states: "'Fancy' may suggest positive chemotherapy, but 'Fact' emphasizes that 'cure' can only be obtained when urinary tract obstructions have been properly corrected."

\* \* \* \* \*

### Minor Eye Signs in Migraine

E. Charles Kunkle MD and W. Banks Anderson MD, Duke University Medical Center, Durham, N. C. Significance of Minor Eye Signs in Headache of Migraine Type. Arch Opth 65:504-508, April 1961.

Ocular manifestations of migraine in its various forms may be obvious or subtle. Although still of unclear mechanism, best known of the major features are the visual prodromes—scotomas and scintillations—of classic migraine. Rare, but no less impressive, is occurrence of temporary or persisting paresis of an extraocular muscle as part of the "ophthalmoplegic" migraine.

The present report concerns certain less dramatic eye signs, all representing additional clues to the nature of migraine. These minor manifestations are particularly prominent in association with headache occurring in cluster pattern, a form of vascular headache described in this century under many different names. It affects mainly adult males and consists of bouts of high intensity pain of relatively brief duration, rarely lasting over 2 hours. It is almost consistently unilateral and anterior in location, usually surrounding the orbit. Most characteristic is its occurrence in bouts of headaches in rapid succession, often one or more each day or night for several days or weeks, followed by remissions lasting several weeks, months, or years. Changing clinical patterns in many instances of cluster headache and existence of transitional forms strongly suggest that it is a variant of migraine. Indirect evidence gained from pharmacologic and other bedside tests indicates that dilation of either intracranial or extracranial arteries contributes to the pain.

Ocular features of cluster headache have been analyzed in 98 patients clearly afflicted with this disorder; of these, 88 were males. The age of onset of the headache varied from 17 to 53 years. Observations here reported can be grouped under two tentative headings.

Parasympathetic Discharge. Common accompaniments of an attack—observed directly or recorded by history in 75% of the group—were injection of the bulbar conjunctiva and excessive lacrimation, usually combined with nasal congestion, all largely or entirely restricted to the side of the pain. The nasal symptom in most instances clearly preceded tearing and included a

sensation of swelling of the turbinates. Rhinorrhea, possibly due in part to the outpouring of tears, was a later and inconstant feature. Enlargement of the ipsilateral superficial temporal artery was noted in a few patients. These signs indicate activity of the secretory and vasodilator nerves to the globe, lacrimal gland, and nasal mucosa traveling from the brain stem by way of the seventh cranial nerve and its greater superficial petrosal branch. Clinical and experimental data, together with previously reported detection of an acetylcholine-like substance in the cerebrospinal fluid during cluster headache in some patients, suggest an integrated parasympathetic discharge largely involving the seventh nerve. Two patients, moreover, were found to have a striking bradycardia during the headache with rates of 38 to 42 and 50 per minute.

Of particular interest were 7 of the 98 patients who developed mild to moderate miosis during attacks of headache. It is possible that such temporary miosis results from activity of pupilloconstrictor fibers of the third cranial nerve in an extension of the parasympathetic discharge within the brain stem as postulated above.

Sympathetic Paralysis. In this group the pupil and lid signs were present both during and between headaches. Six patients had miosis plus ptosis on the side of headache and 2 had miosis alone. These persisting eye signs were so trivial in many that the exact date of their appearance usually remained undetermined, although they followed onset of headaches in all but one individual. Pharmacologic tests with eye drops added to the evidence for sympathetic paresis. In all patients sweating over the face and limbs was unimpaired and ocular tension was grossly normal.

Such evidence of chronic partial ocular sympathetic paresis, allied to Raeder's "paratrigeminal" syndrome, requires some explanation other than that of a recurrent neural discharge. It is tentatively postulated that the signs denote damage to sympathetic fibers in the adventitia of the internal carotid artery as the result of repeated swelling of this vessel in the headache attacks. The responsible lesion may be high, in or close to the carotid siphon, accounting for the locus of the pain and the sparing of fibers serving sweat glands. This same segment of the internal carotid artery has long been held to blame for the extraocular palsies of ophthalmoplegic migraine, usually temporary, rarely permanent. In a few instances, an arteriogram during the paralytic phase has shown apparent narrowing of the distal portion of the internal carotid, tentatively interpreted as a sign of edema of the vessel wall subsequent to its initial dilation.

These separate observations pose a particularly troublesome question: Why should swelling and edema of the internal carotid in a migraine attack lead in some patients to injury to adjacent sympathetic fibers and in other patients to involvement of cranial nerves to extraocular muscles? Perhaps in those with sympathetic paresis the carotid swelling is mainly proximal to the cavernous sinus, whereas in those with ophthalmoplegia it is higher, chiefly intracavernous. Minor hidden variations in the anatomic arrangements of this general area might also account for these differences. In either event it is conceivable that an additional factor in the nerve fiber damage is the noxious



metabolite, recently termed "neurokinin," found in the vicinity of the dilated artery in certain other forms of migraine. Proof of this association is unavailable, nor is it likely ever to be easily accessible, either by chemical or morphologic study. The entire hypothesis must therefore remain highly provisional.

\* \* \* \* \*

### Etiology of Decubitus Ulcers

Michael Kosiak MD, University of Minnesota Medical School, Minneapolis, Minn. Arch Phys Med 42:19-29, January 1961.

Decubitus ulcers are one of the major problems confronting any physician who is called upon to supervise the care of the severely disabled or debilitated patient. Decubitus ulcerations not only prolong morbidity and interfere with the rehabilitation and maintenance of these people, but they also may frequently be implicated as a major contributing factor leading to the patient's demise.

Improved medical, nutritional, and environmental factors have increased the life span of man with the result that more persons than ever before are living to a relatively old age. This presents a problem in that a greater number of patients with cerebral vascular disease, malignancies, and neurologic disorders are being cared for in hospitals and nursing homes.

In spite of the relatively widespread occurrence of ulcerations in the general hospital population and the almost inevitable occurrence in the patient with spinal cord injury, no agreement exists as to the basic underlying cause of the ulceration. This is undoubtedly due to the fact that very little basic research has been done regarding etiology of decubitus ulcers.

Decubital ulcerations are localized areas of cellular necrosis. Normal cell metabolism is dependent on the receipt of nutrients and the elimination of metabolites. Any condition which interferes with this exchange will affect the function of the cell. Because ulcerations occur almost exclusively over bony prominences which have been subjected to excessive pressures for varying lengths of time and because ulcerations almost inevitably heal when pressure is removed, it would appear that ischemia caused by supracapillary pressures is one of the primary factors in production of decubitus ulcers.

To effectively evaluate the physiologic changes which occur as a result of application of pressure, it would appear that three basic problems must be considered.

#### Historical Review

Pressure. It is difficult to evaluate the effect of pressure per se on the physiologic function of the living cell without involving the circulation to the cell. In carefully devised experiments, it has been shown that tissue itself can withstand phenomenal pressure when there is no interference with receipt of nutrients or elimination of waste products of cell metabolism.

Acute Ischemia. Early impetus for investigation of pathologic changes arising from ischemia of voluntary muscle was provided by Volkmann who described the necrosis of muscle due to ischemia induced by compression of the arteries with constrictive dressing. This and other early work devoted attention to chronic lesions with little concern for changes that occur in the first 24 hours. Later observers have considered that early changes leading to ulceration may result from obstruction of arterial circulation, venous obstruction resulting in greatly increased capillary pressure leading to degenerative changes, intrinsic capillary damage, or effects of pressure itself on involved tissue.

Capillary Changes. Tissue fluid interchange at the capillary level has been shown to be affected by a variety of factors intrinsic and extrinsic to the capillary network—arterial blood flow, neurogenic factors, local changes in tissue, and externally applied pressure.

### Experimental Study

This study was undertaken in an attempt to accurately determine the effect of both constant and alternating localized pressure on normal and denervated muscle. Localized pressures were applied over muscular tissue and the relationship between microscopic changes in the muscle and the time and intensity of pressure was noted.

Data have demonstrated the marked susceptibility of tissue to relatively low constant pressures for short periods of time, and somewhat greater resistance to change following application of equal amounts of intermittent pressure. This was true of both normal and denervated tissue.

A definite inverse relationship exists between the amount and duration of pressure which tissue can tolerate before pathologic changes are noted. Intense pressures of short duration are responsible not only for interference and perhaps complete cessation of capillary circulation, but also for changes in the larger vessels with the result that venous thrombi may be found. The presence of venous thrombosis then interferes with the normal reactive hyperemic vasodilation after the pressure is removed, with the result that the tissues continue to remain ischemic.

Pathologic changes following ischemia are logically considered by some to be due to disturbances in capillary circulation; this is as evidenced by changes noted after a single application of constant pressure slightly greater than capillary pressure. Presence of edema and cellular infiltration 24 hours after application of moderate pressure would indicate moderate changes in capillary permeability probably due to capillary membrane ischemia. Increasing the degree or duration of ischemia not only increases the changes in membrane permeability but interferes with cellular metabolism to such a degree as to produce cellular necrosis and inflammatory reaction in the muscle tissue. Again, similar weight of evidence indicates that pathologic changes are due to ischemia from arterial obstruction.

Regarding the importance of a neurotrophic factor as a primary cause of ulceration, there is little basic evidence to lend support to this theory.



Absence of sensation in patients with spinal cord or peripheral nerve injuries may be rightly considered to be the factor in pressure changes in such patients because the patient does not experience the discomfort of continuing pressure.

Other contributing factors in production of ulcerations include metabolic deficits which manifest themselves following trauma or prolonged immobilization, edema of tissues, and anemia.

Because of microscopic degenerative changes which result from application of only relatively low pressure for short periods of time, it becomes apparent that degenerative reaction and recovery from these changes is probably taking place simultaneously. Where tissue is subjected to pressures for only short periods of time, the normal reactive hyperemic response partially compensates for the temporary ischemia with the result that the tissue does not undergo morphologic degeneration.

Clinically, even when adequate nursing care is available, the incapacitated person is rarely turned oftener than once every 2 hours; many bed-ridden patients develop ischemic ulcers on such a regimen. This would indicate that the damage produced during the time that the pressure was being exerted was not completely repaired during the intervals when the area was free of pressure. Little difficulty would arise if a person's weight were equally distributed over the entire surface of the body available for weight bearing when in the recumbent position. However, in an ordinary bed, the weight of the body is borne primarily over certain bony prominences whose combined area is very small with the result that the pressures over these areas undoubtedly exceed capillary pressure.

The weight-bearing area available to the patient is considerably less when sitting than when supine. We have shown that even with a 2-inch foam rubber cushion, average pressures under the ischial tuberosities and the surrounding area generally exceed 150 mm Hg. Cut-out areas intended to decrease or eliminate pressures over the bony tuberosities only tend to divert more of the weight bearing to the surrounding area. Increasing the thickness of the cushioning to as much as 6 inches of foam rubber appears to have had little effect on the occurrence of ulceration in the wheel-chair patient. Conversely, the more sophisticated, well-informed paraplegic with good upper extremities can remain relatively free of ischemic ulcers by conscientious weight shifting several times each hour regardless of the seat padding which is used.

Since it is impossible to completely eliminate all pressure for a long period of time, it becomes imperative that the pressure be completely eliminated at frequent intervals in order to allow circulation to the ischemic tissues.

\* \* \* \* \*

In every affair consider what precedes and what follows, and then undertake it.

—Epictetus

### Intermittent Vertebral Artery Compression

Samuel R. Powers Jr MD, Thomas M. Drislane MD, and Stuart Nevins MD, Albany Medical College of Union University, Albany, N. Y. Intermittent Vertebral Artery Compression: A New Syndrome. *Surgery* 49:257-264, February 1961.

In 1959, Cate and Scott reported the relief of symptoms of arterial insufficiency of the basilar system after endarterectomy of the proximal portion of one vertebral artery. Occurrence of a similar symptom complex consisting of vertigo, syncope, tinnitus, and deafness in a large number of patients without evidence of arteriosclerosis prompted an investigation into other possible causes for vertebral artery insufficiency. This report is concerned with certain congenital anomalies of the subclavian-vertebral axis which may result in intermittent vertebral artery compression.

Twenty-eight patients who presented the chief complaint of episodic vertigo with or without other symptoms referable to brain-stem ischemia were studied. In 17 of these cases the vertebral artery arose from the subclavian artery in common with, or at the level of, the thyrocervical trunk. In 4 other patients the left vertebral artery arose directly from the aortic arch and then entered the vertebral canal at the level of the fourth cervical vertebra. All 21 patients have improved or have been completely relieved of their symptoms after surgical treatment. Seven of the 28 patients showed atherosclerotic disease involving the vertebral-subclavian junction.

#### Clinical Syndrome

Symptomatology of the syndrome of intermittent vertebral artery compression differs from that in arteriosclerotic occlusive disease insofar as the former syndrome is episodic in nature with periods of complete freedom of symptoms alternating with the almost explosive onset of true vertigo.

Although the time of onset of attacks is apparently unpredictable, precipitating factors are either emotional tension or rotation and extension of the head. In either situation the neck muscles, including the scalenus anticus, are drawn tightly over the thyrocervical trunk and subclavian arteries which produces compression of these vessels against the proximal vertebral artery.

Symptoms may vary from simple vertigo to the more complex picture of basilar artery insufficiency. The symptoms of the latter include diminished hearing of the central type with a considerable perceptive component frequently associated with the phenomenon of recruitment. Tinnitus, when present, may persist after vertigo has ceased, and in some patients may be continuous.

Headache is common and usually of the tension type, predominately unilateral and situated in the supraorbital or parieto-occipital area. In more severe cases the pain—facial in location—is a deep-seated ache characteristic of visceral rather than somatic neuropathy. Gastrointestinal symptoms consisting of nausea, vomiting, and explosive diarrhea may accompany the episodes of



vertigo. These symptoms may result from a spread of excitation from the vestibular nucleus to the adjacent vagal centers.

Visual disturbances may be present and are of the perceptive type. The patient may complain of inability to sustain prolonged periods of visual concentration or hallucination of flashing lights or horizontal lines. These symptoms arising from ischemia of the occipital lobe result from insufficiency of the posterior cerebral artery, a branch of the basilar system, and may occur in the presence of normal internal carotid arteries.

Paresthesia, numbness, and coldness of the upper extremity on the affected side are frequently present and are also episodic, usually in association with the symptoms of cerebral ischemia. A definite vasospastic element may exist with rapid changes in color, temperature, and sweating in response to thermal or emotional stimuli. These symptoms are an important diagnostic feature of the syndrome of intermittent vertebral artery compression since they are associated with the physical signs of subclavian artery compression by the scalenus anticus muscle.

Physical findings during asymptomatic intervals between attacks may be limited to those seen in the scalenus anticus syndrome. Diminution or obliteration of the radial pulse usually occurs with the Adson maneuver, although in many instances an oscillometer is required to be certain that a real change is obtained. Great care must be taken not to place the arm in hyperabduction since many apparently normal individuals show a decreased pulse amplitude with the arm in this position.

Performance of the Adson maneuver may precipitate an attack of vertigo. We believe occurrence of such an attack during rotation and hyperextension of the neck to be diagnostic of the vertebral compression syndrome.

A supraclavicular bruit was present in about one-third of our patients, although in some it could be demonstrated only with changes in the position of the head. The bruit can be either systolic or continuous and is usually transmitted along the subclavian artery rather than upward into the neck.

Episodic symptoms of vertebral artery insufficiency, evidence of a scalenus anticus compression syndrome, and a supraclavicular bruit comprise the cardinal clinical features of this syndrome.

### Diagnosis and Treatment

Anatomic diagnosis of an anomalous origin of one or both vertebral arteries can be made from arteriographic studies. Audiograms may be of value; this examination was carried out in all patients reported, with approximately two-thirds showing significant hearing loss of the nerve type. Because of the blood supply it is possible for either the organ of Corti, or the cochlear nerve or brain stem to become ischemic with vertebral artery insufficiency leading to deafness.

Circulatory studies of both arms were carried out before and after surgery in most patients. A grossly abnormal plethysmograph tracing was obtained in 19 of 20 patients studied.

Symptoms of cerebral ischemia in this syndrome are similar to those seen in other diseases involving the brain stem, such as cerebellopontile angle tumors and aneurysms of the basilar artery. For this reason, a rather elaborate study is justified in any patient who complains of vertigo. Each of our patients was subjected to spinal puncture, caloric testing, audiometric examination, and in some cases, an electroencephalogram. In several instances, roentgenograms of the skull were also taken and pneumoencephalography performed. Every patient was then carefully evaluated by the neurologist before arteriographic studies were begun.

Treatment is accomplished by effectively freeing the anomalous artery from obstruction or compression by muscles or membranes.

#### Comment

The exact mechanism by which vertebral artery compression leads to the symptoms of vertigo, tinnitus, and deafness is speculative. Ischemia of the labyrinth might occur since the latter is supplied by the internal auditory artery which may occasionally rise directly from the vertebral before it joins to form the basilar. It would, therefore, be necessary to postulate an additional intracranial anomaly to account for the predominately unilateral symptoms of this syndrome.

A more acceptable explanation is that unilateral vertebral compression produces a certain degree of ischemia throughout the brain stem and in addition there is vasoconstriction of the internal auditory artery. Sympathetic fibers from the cervical ganglion pass directly in the sheath of the vertebral artery and accompany this vessel and its branches. Mechanical compression of the vertebral artery and its plexus could, therefore, result in spasm of the internal auditory artery in much the same way that compression of the subclavian artery and brachial plexus by the scalenus anticus muscle results in vasospastic disorders of the hand.

We have noted that the nerve fibers from the cervical plexes to the vertebral artery are invariably severed during surgical mobilization of this vessel and may be important in obtaining relief of symptoms. This concept of general brain stem ischemia plus spasm of the terminal branches of the vertebral artery would also explain the associated symptoms of nausea and vomiting as due to a direct effect on the vagal nucleus. On the basis of this hypothesis, we now include stripping of the vertebral plexus as a part of the operative treatment.

\* \* \* \* \*

Whether my observations and opinions be disproved or supported, I shall be equally satisfied. Truth is the prize aimed for; and, in the contest, there is at least this consolation, that all the competitors may share equally the good attained.

—Dominic John Corrigan



### Changing Spectrum of Nocardiosis

John F. Murray, Sydney M. Finegold, Seymour Froman, and Drake W. Will, University of California Medical Center, Los Angeles, Calif. The Changing Spectrum of Nocardiosis—A Review and Presentation of Nine Cases. *Amer Rev Resp Dis* 83: 315-330, March 1961.

Nocardiosis is a localized or disseminated infection which may involve all tissues and is produced by members of the genus *Nocardia*. Delays in early diagnosis and treatment of nocardiosis are often due to confusion with pulmonary tuberculosis, systemic mycoses, and closely related actinomycosis. In the laboratory, cultures may be discarded too early or mistaken for saprophytic acid-fast bacilli. Correct diagnosis is imperative because nocardiosis is susceptible to sulfonamide therapy, but refractory to chemotherapeutic regimens now in use for tuberculous, fungous, and most bacterial infections.

Once considered to be extremely rare, both localized and disseminated forms of nocardiosis appear to be increasing in incidence. In contradistinction to tuberculosis and other fungous infections, little attention has been given to nocardiosis as one of the penalties of sustained steroid therapy as well as its recognition as an infection with a probable association with neoplastic disorders.

#### Historical Review

The first description of disease produced by aerobic actinomycetes was written in 1888 by Nocard who had studied farcy (glanders) in cattle. Eppinger's description of so-called pseudotuberculosis produced by similar aerobic branching organisms in man is considered to be the first report of human nocardiosis. Since that time, 179 cases of nocardiosis have been reported in the literature, with an over-all mortality of 54% (before sulfonamide, 75%).

The great majority of cases of nocardiosis are pulmonary; 31% remain confined to the respiratory tract with no evidence of dissemination or multiple system involvement. It is not commonly recognized that generalized or disseminated nocardiosis is almost as frequent as pure pulmonary disease (30%). Skin and subcutaneous tissue involvement occurs in 14.5%; involvement of most other tissues and organs has been reported at one time or another. Occasionally organisms are isolated from blood cultures.

Nocardiosis is occasionally associated with other diseases; this association is slowly increasing, particularly in those chronic diseases in which long-term steroid therapy is used. These include: leukemia, Hodgkin's disease, lupus erythematosus, asthma, pemphigus vulgaris, and pulmonary alveolar proteinosis.

Diagnosis is made by bacteriologic culture; satisfactory serologic tests have not been developed. Results of both experimental and clinical studies lead to the conclusion that in vitro drug-susceptibility tests are not accurate guides to the therapy of nocardiosis. Sulfonamides are the only drugs that have proved effective; results with antimicrobials are variable and conflicting. An

indispensable aspect of treatment of nocardiosis is adequate surgical drainage of purulent collections and excision of all necrotic tissue.

### Current Observations

The writers' experiences with 9 cases of nocardiosis are presented to illustrate the vagaries of the disease; certain conclusions and observations may be made.

There is no pathognomonic clinical syndrome in nocardiosis. Any tissue may be involved, which leads the writers to propose the following classification:

- I. Acute
  - A. Focal abscess (any tissue)
  - B. Bronchopneumonia
    - 1. With or without abscess
    - 2. Locally invasive (pleura, chest wall)
- II. Chronic
  - A. Abscess (usually pulmonary)
  - B. Fibronodular disease

Even though the lung is most commonly affected, the extent and type of pulmonary disease varies considerably including lung abscess, necrotizing pneumonia, progressive fibrosis, empyema, and fistula.

In addition to local invasiveness, the tendency for vascular involvement with metastatic abscess formation is recorded, with a propensity in several cases for metastatic nocardiosis to involve the central nervous system. Although the brain was the most frequent site of extrathoracic nocardiosis in the present series, other lesions were found in the liver and kidneys in one case, and in the spleen in another.

Although nocardiosis was not considered in any patient on admission, the diagnosis was subsequently made by recovery of the organism from sputum, thoracentesis fluid, or abscess drainage. In order to hasten and improve the diagnosis of nocardiosis, it is imperative that appropriate cultural technics be used.

In view of the apparent increasing incidence of nocardiosis, it is possible that an endogenous source of *Nocardia* is important as a source of disease. Rather than an increased prevalence or greater virulence of nocardial organisms explaining a higher incidence of the disease, it is the writers' contention that a changing status of the host renders him more susceptible to infection. There is probably nothing unique in the ability of *Nocardia* to produce disease in this setting; it is one of many organisms that take advantage of alterations in the host's defenses and normal bacterial flora to thrive.

A defect in the immune processes may occur in many different diseases. There is also a convincing amount of evidence to support the notion that lowered immunity to infections results from use of adrenocortical steroid drugs and



many antineoplastic agents. When one or more of these factors exist, bacterial infection commonly occurs requiring prolonged antimicrobial therapy; it is in this setting that secondary infections can readily develop. Mandatory use of corticosteroids, antineoplastic drugs, and antimicrobials is widespread and increasing. A corollary of this practice is that such infections will also increase. This is an additional reason for avoiding unnecessary or indiscriminate use of antimicrobials or steroids.

The prognosis of uncomplicated nocardiosis in the present series depended primarily upon the extent of the infection, the time of recognition of the disease, and effectiveness of chemotherapy and surgical treatment. It is obvious that the prospects of any underlying disorder seriously affect the outcome of nocardiosis.

Diagnosis of nocardiosis has always been difficult due to the variable manifestations of the disease; when the infection occurs with a multicentric systemic disorder, the clinical features are even more complex. Difficulties in distinguishing between tuberculosis, actinomycosis, and nocardiosis have troubled physicians for many years.

Awareness of the possibility of nocardiosis and obtaining appropriate cultures are the most important aspects favoring early diagnosis. Nocardial invasion should be considered, especially when underlying circumstances enhance secondary infection. Vigorous chemotherapy and surgical treatment can eradicate the infection and should lead to a successful outcome in many patients.

\* \* \* \* \*

#### Complications of Viral Hepatitis

Russell J. Blattner MD. Comments on Current Literature: Complications of Viral Hepatitis. J Pediat 58:596-599, April 1961.

The possible occurrence of "autoimmune" reactions against human tissue antigens in certain acute and chronic diseases has attracted increasing attention in recent years. In 1956, Bearn, Kunkel, and Slater described a condition accompanying, or associated with, chronic hepatic disease. The syndrome occurred predominantly in young girls and was characterized clinically by amenorrhea, obesity, striae, arthralgia, skin rashes, with obscure febrile episodes, and evidence of moderate or severe liver damage. Most of the patients showed a marked elevation in serum gamma globulin. Biopsy specimens obtained early in the course of the disease revealed infiltration of the periportal areas with an inflammatory cell exudate made up largely of plasma cells. In a few of these cases the condition was thought to be the result of anicteric viral hepatitis, but in most instances the etiology was considered unknown. Kunkel and his associates stressed the insidious onset of the disease and absence of a history of acute infectious hepatitis.

Subsequently, Page and Good described clinical management, including steroid therapy, of a number of patients, both boys and girls, showing this



syndrome. They referred to the condition as a "plasma-cell hepatitis," emphasizing the presence of the plasma cell infiltrate in the liver, and called special attention to the related hypergammaglobulinemia. In their patients, a history of preceding infectious hepatitis was common. Among those without a specific history of hepatitis, nonicteric undiagnosed hepatitis—which is thought to occur frequently in young children—could have occurred. Evidence of generalized vascular disease in their patients, along with plasmacytosis and markedly elevated gamma globulin levels, led Page and Good to "consider the possibility that this disease is an expression of hypersensitivity."

The concept of liver damage on an autoimmune basis is not a new one, and possible sensitivity reactions include that of response to "an exogenous antigen persisting in the liver and the sensitivity of the patient to his own liver tissue."

As early as 1944, Eaton and his colleagues were able to identify a complement-fixing antibody against human liver in the serum of patients with acute hepatitis and suggested that an autoimmune mechanism might account in some instances for late reactions to infectious hepatitis. More recently, Gajdusek showed the presence of a complement-fixing antibody against desoxy-ribose-nucleoprotein in patients with chronic hepatitis, lupus erythematosus, and acute glomerulonephritis. Similarly, Mackay and his co-workers found 5 patients with chronic hepatitis, all of whom had elevated gamma globulin levels and sedimentation rates. Mackay offered the explanation that these patients represented cases of autoimmune hepatitis resulting from changed antigenicity of liver cells by viral or nutritional injury.

Six of the 10 patients observed by Page and Good were considered to be representative of the plasma cell hepatitis syndrome: "Extreme hypergammaglobulinemia, severe chronic hepatitis, and manifestations of generalized vascular disease such as skin rash, arthritis; and characterized histologically by extensive plasma cell infiltration of the liver." In these 6 patients, cortisone therapy was effective in suppressing all clinical manifestations of the disease and most of the laboratory indications. In addition, improvement in the histologic appearance of the liver was evidenced by biopsy before and during therapy. In some patients, however, serious side effects of steroid therapy occurred. Four patients with chronic hepatitis, moderate hypergammaglobulinemia, and manifestations similar to those of plasma cell hepatitis also received cortisone therapy. In this group, however, the results were not encouraging.

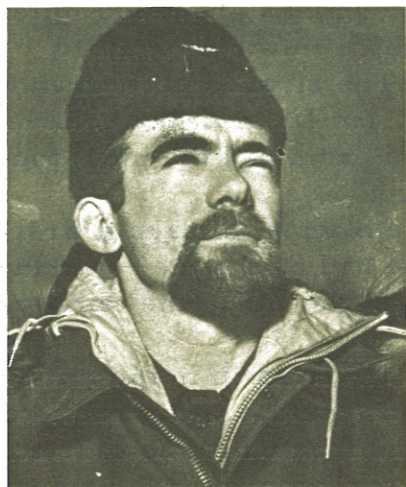
In a discussion of the plasma cell hepatitis syndrome, Page and Good point out that each of the etiologic concepts offered in recent years is logical and tenable: autoimmune disease of the liver, hepatic damage occurring during the course of acute virus hepatitis, possibly as a result of hypersensitivity to the virus; liver damage in chronic hepatitis; and postnecrotic cirrhosis following virus hepatitis resulting from actual persistence of virus in the tissue. In the latter hypothesis, the plasmacytosis and elevated gamma globulin levels could be regarded as the result of prolonged antigenic stimulation by the virus.

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Cold Country "Doc"

Scot MacDonald JOC USN, All Hands, May 1961.



LT A. Michael Pardue, MC, USN

"Whoever said there are no bacteria in Antarctica is just plain wrong." That's the word from a Navy physician who recently returned from the Antarctic. He was one of 11 physicians and 2 dentists serving in the scientific support operation.

LT A. Michael Pardue is the flight surgeon assigned to Air Development Squadron VX-6, and he's firmly convinced that wherever there are people or animals, there are bound to be bacteria. The Antarctic, which some sources have indicated is too cold to sustain bacteria, is no exception.

"For example," cites the doctor, "men bring colds to the Antarctic from warmer climates. The cold germs spread from man to man, and before you know it the infected 80% of newcomers have infected 20% of the previous wintering party."

When the group returned to the U.S., the doctor was able to provide a first-hand account of cold weather medicine and life at the bottom of the world. Before he headed south, he saw to it that the personnel were physically and mentally qualified for the isolated duty that faced them. Each man had to pass a physical examination more strenuous than those given submariners.

Dr. Pardue arrived on the Antarctic ice landing strip at McMurdo Sound in a VX-6 Super Constellation. There he established a canvas and wood medical clinic, small and barely equipped to meet minimum standards. Serious cases were to be referred to the main camp dispensary at McMurdo. He remained on the landing strip for the entire summer tour with only a two-week break and an occasional trip to McMurdo. The runways are carved out of the bay extension of the Spain-sized Ross Ice Shelf, the largest mass of floating ice in the world. From there, VX-6 planes flew to outlying stations in the middle of Marie Byrd Land and at the geographic South Pole.

During the peak of the summer support operation the population at McMurdo exploded to nearly 700 including scientists and military. Nearly half the military were quartered in huts on the ice strip—thus the need for Dr. Pardue and his clinic.

The most common medical ailment the cold weather doctor treated was dry cough. Most of the men who complained of the cough came down with it during their first few days on the ice. The cough was caused by the extreme dryness of the Antarctic climate and was aggravated by overheated huts and a general increase in smoking. The ailment usually subsided in about two weeks, as soon as the men became accustomed to the climate.

The doctor's first emergency situation came in late October when a WV-2 aircraft crashed at McMurdo while attempting to land. The 12 men aboard

the plane were given immediate first aid treatment. Four were listed in critical condition. They were flown by VX-6 helicopter to McMurdo's dispensary where more complete medical facilities were available.

In November, the doctor was confronted with another emergency when a ski-equipped R4D Skytrain crashed in the Horlick Mountains about 300 miles north of the Pole. Dr. Pardue boarded an aircraft which flew him to Byrd Station and he continued from there to the Horlicks. The stranded crewmen of the crashed aircraft had radioed they were uninjured, but Dr. Pardue believed that the men might have minimized their injuries. He examined the men, found them shaken but fit, and returned to his vigil on the runways.

Just before Christmas, the doctor flew to Pole Station to make a first-hand check on the effect cold weather and high altitude had on the men working there. Pole Station is 9200 feet above sea level and, at the time, the temperature was 20 degrees below zero.

All in all, Dr. Pardue treated few injuries at the airstrip in the Antarctic, and ascribed it to the fact that the men selected for the operation had intensive pre-deployment training. He was surprised at the limited cases of frost-bite and snow blindness, but expressed concern over carbon monoxide poisoning, an ever-present danger in Antarctic expeditions.

Commenting on Antarctic food, the doctor said that the men are well fed; their daily ration is one and one-half times as much as the average seagoing sailor's. Antarctic workers need the extra food to provide the energy needed in cold weather.

On the subject of bathing, once a week is a rule of thumb in the Antarctic. The unwritten order is routinely observed in an effort to conserve water which must be melted from snow. Also, a solid medical reason is that the climate dries the skin. Excessive use of soap dries the skin even more.

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### Life Expectancy Following Retirement

A pessimistic attitude may be held by many in relation to years of life to be enjoyed following retirement from the Navy. One team in Japan, making a Standard Career Appraisal Presentation, states that they frequently hear the rejoinder: "But the average guy doesn't live long after he retires from the Navy." Then a story about the man who retired from the Navy one day and died the next usually follows, giving the impression that perhaps Navy life is not considered good preparation for retirement. Troubled by these comments for which they had no satisfactory answer, the NavCAT team sought information from a local Medical officer; he in turn suggested they solicit the Medical News Letter for information or statistics to aid in combating this apparently not uncommon belief.

Specific facts of longevity following retirement either have been not available for study or not sufficiently analyzed for publication by the Medical Department of the Navy, nor by the Bureau of Supplies and Accounts. The



latter might be expected to be particularly interested in the "cost" of such longevity. The Department of Defense, also fully aware of the interest and value of such a study, is preparing to process myriad figures when sufficiently available, with expectations of arriving at some concrete conclusions from the overall viewpoint of the Armed Forces. However, the Medical Statistics Division of the Bureau of Medicine and Surgery, after exploring several avenues of information, discovered the accompanying table which was presented in Vital Statistics of the United States, 1958, Section 5, Life Tables, prepared by the U. S. Department of Health, Education, and Welfare.

AVERAGE REMAINING LIFETIME,\* U. S. Males: 1958

Age Group (Years)	Average Remaining Lifetime (Years)		
	Total Male	White Male	Nonwhite Male
35-40	35.8	36.0	32.0
40-45	31.1	31.5	28.0
45-50	26.8	27.1	24.1
50-55	22.7	22.9	20.5
55-60	19.0	19.2	17.3
60-65	15.6	15.7	14.5
65-70	12.6	12.7	12.1
70-75	10.1	10.1	10.9
75-80	8.0	7.8	9.9
80-85	6.0	5.8	8.6
85 and over	4.7	4.5	7.7

\*Average number of years of life remaining at beginning of age interval.

Although the table considers the entire population of the United States (regardless of state of health) it is only reasonable to assume that it may be broadly applied to the Navy population. Actually, the average remaining lifetime for the man who retires without physical disability should be considerably greater than that in the table because the Navy man represents a physically select group. Furthermore, he has had good medical attention provided him throughout his years of service. Therefore, he should have a greater life expectancy at any given age than Mr. Average Citizen who did not have available, or take advantage of, expert medical attention, and who has not profited from the carefully worked out preventive medicine programs of the Navy.

On the other hand, an individual with physical disability retirement may have a shorter remaining lifetime, with great variation depending on the cause and degree of disability.

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Navy Flight Surgeons Receive  
Three of Aerospace Medical Association Awards

Three Navy Flight Surgeons were selected for recognition in conjunction with the annual meeting of the Aerospace Medical Association held in Chicago 23-27 April 1961. The Association presents several awards each year for outstanding achievements. This year, CAPT Ashton Graybiel MC USN received the Eric Liljengrantz Award for outstanding basic research accomplishments in problems relating to acceleration and altitude. CAPT Philip B. Phillips MC USN was presented the Raymond F. Longacre Award for outstanding accomplishments in the psychologic and psychiatric aspects of Aviation Medicine. CAPT Carl E. Wilbur MC USN was honored with the Harry G. Moseley Award for his material contributions to flying safety.

At the Honors Night Banquet on 26 April, RADM J. L. Holland MC USN, Commanding Officer of the Naval Aviation Medical Center, Pensacola, Fla., accepted the presidency of the Association for the coming year. The General Chairman for the annual meeting to be held next year will be CAPT F. B. Voris MC USN, Director, Aviation Medicine Technical Division, and Head, Aviation Medicine Systems Requirements Branch, BuMed.

Navy Flight Surgeons serving in various capacities for the 32nd annual meeting of the Association, just closed, were: CAPT M. H. Goodwin, Assistant Chief of the Bureau of Medicine and Surgery for Aviation Medicine, and Director, Aviation Medicine Operations Division, represented the Surgeon General; CAPT A. P. Rush MC USN, Head, Aviation Medicine Training Branch, BuMed, served as Coordinator of Navy Scientific Exhibits; CAPT J. P. Pollard MC USN, Director, Astronautical Division, BuMed, and Special Assistant for Medical and Allied Sciences, Office of Naval Research, acted as Chairman of the Education and Training Committee and was a member of the Executive Council; CAPT Voris, next year's General Chairman, was Chairman of this year's Membership and Credentials Committee and served as Chairman of the session, Escape from High Performance Aircraft.

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Armed Services Orthopedic Seminar

The Air Force Medical Service will act as host for the Third Armed Forces Orthopedic Seminar which will be held at the U. S. Air Force Hospital Lackland, Lackland Air Force Base, Texas, 20 - 22 September 1961.

Material to be presented is especially selected to cover the problems of common concern most frequently encountered by orthopedic surgeons of the Armed Forces. Presentations will also be made of results of clinical research projects jointly carried out by the three services during the preceding year. All Orthopedic Surgeons and Orthopedic Residents on active duty in the Armed Services are eligible to attend. Eligible and interested Navy Medical officers should forward requests to Chief, Bureau of Medicine and Surgery, via chain of command,



in accordance with BUMED INSTRUCTION 1520.8, at least 8 weeks prior to commencement of the seminar. Travel and per diem orders chargeable against Bureau funds will be authorized for attendance of selected officers and residents contingent upon availability of funds.

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American Board Examinations -  
Obstetrics and Gynecology

Applications for certification in the American Board of Obstetrics and Gynecology, new and reopened, 1962 Part I examinations, and requests for reexamination in Part II are now being accepted. All candidates are urged to make such application at the earliest possible date. Deadline date for receipt of applications is 1 August 1961.

Candidates for admission to the Examinations are required to submit with their application a list of all patients admitted to the hospitals where they practice. This and other requirements are described in detail in the current Bulletin of the Board which may be obtained from Robert L. Faulkner, MD, Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

After 1 July 1962, the American Board of Obstetrics and Gynecology will require a minimum of three (3) full years of approved progressive Residency Training to fulfill the requirements for admission to examination. After that date, training by Preceptorship will no longer be acceptable.

The Board particularly requests all Diplomates to notify the office of the Secretary as soon as possible of any change of address.

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From the Note Book

NAS Pensacola, Host to Naval Aviators. Naval Flight Surgeons—active and inactive—are advised that from 6 to 11 June 1961, the Naval Air Station, Pensacola, Fla., is playing host to naval aviators from all over the country in connection with celebration of the 50th Anniversary of Naval Aviation. Flight Surgeons who happen to be vacationing or otherwise in that area at the time may well find many old friends. The Naval School of Aviation Medicine will participate in activities.

Esophagoscopy by and for the Internist. This paper presents a review of esophagoscopy performed in 1000 patients in the past 8 years on the medical service of two hospitals. The author believes that sufficient experience has been obtained by enough workers in the field of esophagoscopy to indicate the safety and value of the procedure and the necessity for a broader teaching program to train more internists with gastrointestinal interests in this technic. (I. Brick, Amer J Med Sci, March 1961)

Treatment of Carbon Monoxide Poisoning. A Preliminary Communication from University Department of Surgery, Western Infirmary, Glasgow, Scotland describes correction of anoxia of carbon-monoxide poisoning by pressurization to two atmospheres of oxygen. By this means the cellular anoxia is immediately corrected, and furthermore it follows that more hemoglobin which can combine with oxygen will be liberated from carboxyhemoglobin. The carbon monoxide is excreted through the lungs and normal oxygen transport by the blood is restored. Two cases showing prompt recovery following pressurization are described. (The Lancet, October 22, 1960)

Anticoagulant Therapy in Coronary Heart Disease. Studying selected patients in Oslo, Norway over a two and a half-year period, the authors concluded that patients with angina pectoris, with or without previous myocardial infarction, should be given long-term anticoagulant therapy regardless of the severity of the symptoms. (C. Borchgrevink, Acta Med Scand, Sup 359, Vol 168, 1960)

Dangers of Phenacetin. The widely-held assumption that the combination of phenacetin, aspirin, and caffeine has greater analgesic effectiveness than the equivalent dose of aspirin alone has never been supported by carefully controlled trials. Despite the difficulty of distinguishing the effects of acetophenetidin from those of other drugs given simultaneously, it is clear that doses of a gram or more taken daily for months or years have occasionally caused methemoglobinemia, sulfhemoglobinemia, and hemolytic anemia. Recent clinical and pathologic studies also indicate that excessive use of phenacetin can cause severe renal injury. It would seem prudent for physicians and dentists to restrict refill privileges on phenacetin prescriptions; and labels on over-the-counter products containing the drug should give clear and emphatic warnings against excessive use. (The Medical Letter on Drugs and Therapeutics, March 17, 1961)

Amebiasis in U. S. and Canada. A study was made of the prevalence of *Entamoeba histolytica* as reported in 166 surveys on nearly a quarter of a million persons in the United States and Canada. Previous estimates of 10 to 20% prevalence of amebiasis in the U. S. were found to be much too high because of inclusion of *E. hartmanni* and because the majority of surveys were done on groups expected to show a high prevalence of the disease. The present estimate is that about 1% or less of the population of Alaska and Canada and less than 5% of the U. S. population harbor *E. histolytica*. (R. Burrows, Amer J Trop Med, March 1961)

Spray Surgical Dressing. Rezifilm, a sterile spray of methacrylate resin, was employed as a postoperative dressing following dermatologic surgical procedures on ambulatory office patients. It was found to be excellent for both unsutured and sutured wounds. A single application remains in place for 7 to 10 days and prevents infection of the wound during this period. (N. Kanof and S. Blau, Arch Derm, March 1961)



**DENTAL****SECTION**

### Bruxism and Chronic Headache

Ragnar Berlin and Leopold Dessner, Central County Hospital, Falköping, Sweden. Lancet No. 7145:289-291, August 6, 1960; abstr in Dental Abstracts 6:248-249, April 1961.

Bruxism is most common during sleep but may be noticed during the day when the patient is concentrating on some trying work, particularly when he is upset or irritated. The hypertonic state of the muscles when the jaws are pressed together, either consciously or unconsciously, soon produces a progressive ischemia, with impairment of oxygenation and accumulation of metabolic products. As a result, the sensory nerve endings are gradually stimulated, causing pain. In addition, the hypertonic muscles pull on the tendons and supporting tissues. All these factors may contribute to the pain which arises in the spastic muscles of the stomatognathic system and is felt as headache.

Bruxism implies long-standing isometric muscle activity leading to a spastic condition within the masticatory muscles. Local factors causing bruxism include various disturbances in occlusion, such as interceptive occlusal contacts, interferences in gliding movements, and closed bites. To elicit bruxism, some additional factor is necessary; this would seem to be psychic. Bruxism is most common in people who are under emotional tension.

For diagnosis, the case history is extremely important. The patient usually wakes up with a headache and often he will admit to experiencing tenderness in the teeth or the masseter and temporal muscles. With the patient's teeth in tight occlusion, often it is possible to palpate hypertrophic and tender masticatory muscles, and often there is tenderness along the zygomatic arch. The lower jaw cannot be relaxed to the normal extent, and the freeway space is reduced to 0 to 1.0 mm. Usually, one can find atypical attrition facets on the teeth, particularly on the cuspids and bicuspid.

The diagnosis of bruxism was made in 62 patients, of whom 51 were women and 11 were men. The aim of treatment is to break the vicious circle by making it impossible for the patient to clench his teeth. This can be done with a "relaxation" appliance designed by Dessner (1959). The purpose is to open the bite anteriorly by inserting a bite plane between the upper and lower incisors, thus increasing the intermaxillary space. The appliance is made of acrylic resin and is worn on the upper jaw. It is retained by a pair of simple clasps. The appliance is intended to be worn during the night and, when

indicated, during the day also. It is worn regularly for at least six months, that is, for at least 4 to 5 months after disappearance of the headache, which usually takes place within four to six weeks.

Of 62 patients for whom appliances were prescribed, 42 (68%) were completely cured in one or two months; 12 (19%) were considerably improved; 6 (10%) were only slightly improved; and 2 (3%) were not improved. Thus the treatment was successful in 87% of the patients. Follow-up study showed that the results remained essentially unchanged for up to three and a half years.

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### Topical Corticosteroids for Relief of Pain

Arthur E. Fry, Robert F. Watkins, and Nilkanth M. Phatak, Dental School, University of Oregon, Portland, Ore. Topical Use of Corticosteroids for the Relief of Pain Sensitivity of Dentin and Pulp. Oral Surg, Oral Med, & Oral Path 13:594-597, May 1960; abstr in Dental Abstracts 6:215, April 1961.

Forty-three teeth with either pulpal exposure or carious invasion were studied and treated with a medication consisting of a small drop of camphorated para-chlorophenol with metacresol acetate to which was added enough prednisolone acetate powder to form a thin paste.

Teeth in which pulpal hyperemia had occurred as a result of restorative interference were treated as follows:

1. When a full crown preparation was involved, the prepared medication was initially applied to the exposed dentin, after which a temporary crown was cemented with zinc oxide-eugenol. In restorative procedures involving inlay or gold foil restorations, the restorative material was removed, medication was applied and the cavity was sealed with zinc oxide-eugenol and zinc oxyphosphate cement.

2. When a vital operative pulpal exposure had occurred under aseptic conditions, the paste was picked up on a small pellet of cotton and placed directly over the exposure to protect the pulp. A thin mix of zinc oxide-eugenol was placed over this without applying pressure. Occasionally, an autoclaved asbestos disk or cigarette paper was placed over the medication, after which the zinc oxide-eugenol was placed and the cavity was sealed with zinc oxyphosphate cement.

3. When patients reported dental pain and roentgenograms indicated the possibility of pulpal exposure if carious dentin were removed completely, the carious dentin was only partially removed, and the paste was placed over the unexcavated carious dentin. The paste was covered with zinc oxide-eugenol and the seal was completed with oxyphosphate cement.

All patients said that their pain hypersensitivity had subsided promptly. When queried 24 hours postoperatively, most patients said pain was absent. The elapsed time between the initial treatment and further rechecks varied



from 4 to 6 weeks. On recall examination, all treated teeth responded normally to vitality tests. All patients were free of symptoms and roentgenograms showed evidence of calcification or the formation of secondary dentin. To date, only one of the 43 treated teeth has had to be extracted.

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Cricothyroidotomy—Slide Lecture  
Available for Loan

Cricothyroidotomy, the fifth in a series of slide lectures prepared by the U. S. Naval Dental School, National Naval Medical Center, Bethesda, Md., is now available for loan on a short-term basis. The study set consists of 38 colored slides (35 mm), a bound narration in lecture form, hand viewer, and carrying case packaged to facilitate use by students, instructors, or professional groups. Ten of the sets are available.

Other slide lectures prepared by the Naval Dental School include: (1) Mouth Preparations for Removable Partial Dentures, (2) Remount Technique for Occlusal Correction of Complete Dentures, (3) Diagnosis for Complete Dentures, and (4) Occlusal Equilibration of Complete Dentures.

Loan of a slide lecture set may be requested by submitting a letter in the following form:

From: \_\_\_\_\_  
(name, rank, full address)

To: Commanding Officer, (Code 7), U. S. Naval Dental School, National Naval Medical Center, Bethesda 14, Md.

Subj: Illustrated lecture, request for loan of

1. It is requested that I be granted the loan of the illustrated lecture \_\_\_\_\_ for approximately 2 weeks.
2. It is requested that the period of the loan commence on, or about, \_\_\_\_\_, 196 , to expire not later than 2 weeks from date of receipt.
3. I will exercise due care in handling and stowing this training material and will return it in the original carton with the enclosed franked address labels attached at the expiration of loan period.

\_\_\_\_\_  
(Signature)

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Russian Medical-Scientific Society  
of Stomatology

At the last meeting of the Russian Medical-Scientific Society of Stomatology in Kiev, May 1959, a program for future dental care of the Russian population sponsored by the government and the dental societies was unanimously adopted. The plan is designed to provide dental services on a three level basis: (1) Treatment of patients who voluntarily visit dental clinics (prosthetic or preventive treatment excluded). (2) Compulsory periodic examinations and treatment at school dental clinics. This service may occasionally be extended to pregnant women, hospitalized patients and members of the armed forces. (3) Systematic preventive procedures to reduce the incidence of dental caries and periodontal disease. This service is free of charge and at present only for children aged 3 to 14 years. Seventy independent dental polyclinics and 2000 new school dental clinics were created for the purpose and as the number of graduate dental students has increased by 300% over that of 1957, the school dental service in all parts of the Soviet Union will be increased and facilities added to those already existing. (Federation Dentaire Internationale News Letter March 1961)

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American Board Certifications

American Board of Oral Surgery

CAPT Robert A. Middleton DC USN

U. S. Naval Hospital, Oakland, Calif.

CDR Guy R. Courage DC USN

U. S. Naval Dental Clinic, Long Beach, Calif.

LCDR Howard S. Kramer Jr DC USN

U. S. Naval Hospital, Charleston, S. C.

American Board of Periodontology

CAPT Robert H. Loving DC USN

U. S. Naval Training Center, Great Lakes, Ill.

LCDR Walter H. Johnson DC USN

U. S. Naval Hospital, San Diego, Calif.

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Personnel and Professional Notes

LT Tibbetts Presents Table Clinic. On 7 May 1961, LT V. R. Tibbetts DC USN, U. S. Naval Station, New Orleans, La., presented a table clinic—Manifestations of Bone Pathosis in the Dental Roentgenogram—before the Louisiana State Dental Association's Annual Meeting held in Lafayette.



Taipei Dental Department in New Quarters. Upon completion of the \$200,000 Station Hospital at the U. S. Naval Support Activity, Taipei, Taiwan, the Dental Department of the facility has moved into a new ten dental operating room wing. The department is staffed by 10 Dental officers and 16 dental technicians and is equipped to provide all authorized dental services for U. S. Armed Forces personnel and their dependents in the Taipei area.

Dedication ceremonies were held on 9 April 1961, with Vice Admiral Roland N. Smoot USN, Taiwan Defense Command, cutting the ribbon to officially open the new hospital. CAPT R. S. Brookings II USN, Commanding Officer, U. S. Naval Support Activity, received the key to the building from a member of the staff of Commander Naval Forces Philippines, CAPT J. M. Raymond CEC USN. A cake-cutting ceremony was performed jointly by CAPT I. G. Edwards DC USN, Senior Dental Officer and CAPT J. W. Firoved MC USN, Senior Medical Officer.

General Anesthesia for Dentistry. General anesthesia for dentistry was discussed by a panel of dentists and physicians at the U. S. Naval Dental School recently. CAPT M. G. Turner DC USN, Acting Commanding Officer, welcomed the panelists. CAPT D. E. Cooksey DC USN, Head of the Oral Surgery Division, served as moderator and presented the panel to the audience of staff, resident, and postgraduate Dental officers, and civilian and military guests.

CAPT J. G. Kurfees MC USN, Chief, Anesthesiology Service, U. S. Naval Hospital, Bethesda, Md., discussed anesthesia for dentistry in a large hospital. Dr. Frank J. Grabill, Washington, D. C., discussed general anesthesia in the private office, and Dr. Robert J. O'Brien, Arlington, Va., described intravenous anesthesia in the office practice of the oral surgeon. A question and answer period closed the meeting.

Philadelphia Meeting. During the recent annual meeting of the Greater Philadelphia County Dental Society, several Dental officers of the U. S. Naval Hospital, Philadelphia participated. CAPT E. A. Gargiulo, Chief of Dental Service, addressed the Dental Assistants of the Delaware Valley Area, discussing: The Duties of the Dental Assistant as to Patients, Office Procedure, Public Relations and the Team Plan Concept. Before the Combined Group of the Philadelphia County Dental Society, CAPT J. F. Bowman presented a table clinic: Complete Denture Impression Procedures; and CAPT E. A. Gargiulo with LTs A. D. Loizeaux and K. W. Besley presented a table clinic: Lines of Incision in Oral Surgery.

Wisconsin State Dental Society. On 26 April 1961, Navy Dental officers participating in the 91st Annual Meeting of the Wisconsin State Dental Society held in Milwaukee, were: Dental Research at Great Lakes, Illinois—CAPT R. B. Wolcott, CDR M. A. Mazzarella, and LT W. R. Shiller; Class V Gold Foil Restoration—CAPT R. B. Wolcott, LT R. N. Draheim, LT B. J. Grothaus, LT R. T. Rydstrom, LT R. F. Wilkin, and LT C. B. Wills.

Rhode Island Dental Society. Dental officers of the U. S. Naval Station, Newport, R. I., sponsored the Spring Meeting of the Rhode Island Dental Society at the Station's Commissioned Officers Mess (Open). CAPT W. H. Snyder DC USN, Senior Dental Officer, presided as Sponsorship Chairman of the professional portion of the meeting which included the following clinics:

CAPT R. F. Erdman—Review of Mass Casualty Treatment as it Applies to Dentistry

CAPT R. V. Peterson—Coil Spring Precision Attachment in Partial Dentures - A Practical Report

CDR A. E. Smith—Occlusal Equilibration in Complete Dentures

LT J. F. Solverson—Practical Endodontic Procedures

Seminar in Japan. The 9th annual Seminar of the American Stomatological Society of Japan was held at Camp Zama, Japan, 20 - 22 April 1961. CAPT Robert D. Wyckoff DC USN, Staff Dental Officer, Commander Naval Forces Japan, and Commanding Officer, U. S. Naval Dental Clinic, Yokosuka, Japan, took part in welcoming the attending Armed Forces Dental officers and outstanding civilian members of the dental profession from the United States and other Pacific areas. Two notable speakers on the program were RADM G. C. Paffenbarger DC USNR who spoke on Clinical Applications of Dental Research, and Dr. Max Shapiro who spoke on Some Considerations of Periodontics.

Dental officers of the U. S. Naval Dental Clinic, Yokosuka, Japan, taking an active part in the 3-day program were:

#### Essays

LT P. L. J. Bradford—Endodontics in Military Practice

LT D. C. Davis (USNR)—Dental Assistants

LT S. J. Hunter (USNR)—Use of Rubber Dam in Dentistry

#### Table Clinics

CAPT L. G. Hopfer—Monoplane Occlusion in Complete Dentures

CAPT J. P. Jones and

LT M. C. Corbett—Denture Characterization

CAPT C. E. Kailer—Rest Preparations for Partial Denture Prosthesis

LT M. S. Jacobs (USNR) and

LT P. W. O'Shields (USNR)—Temporary Acrylic Bridges

LT S. J. Hunter—Audio Analgesia

LT W. J. Toth (USNR)—Sealing Properties of Various Dental Materials

LT A. W. Landel—Use of Amalgam with Low Mercury Alloy Content

LT G. F. Prenier—Amalgam Restorations Reinforced with Pins

CAPT Pridgeon Is Guest Speaker. CAPT C. T. Pridgeon DC USN, Base Dental Officer, Marine Corps Base, Camp Lejeune, N. C., recently was the guest speaker at the monthly meeting of the W. W. Demeritt Pedodontic Study Club at New Bern, N. C. He discussed: The Periodontist Looks into the Infra Bony Pocket with the Pedodontist.



**RESERVE****SECTION**

Active Duty for Training (ACDUTRA)  
During Fiscal Year 1962

Courses representing a portion of Active Duty for Training (ACDUTRA) authorized for fiscal year 1962 are hereby described for information of eligible inactive Medical Department personnel in pay and non-pay programs of the Naval Reserve. Assignment to active duty for training is a matter within the cognizance of the respective Naval District Commandants; interested Naval Reservists should apply to their commandants concerning any of the courses. (Note: Other types of active duty for training available during fiscal year 1962 will be published in succeeding issues of the Medical News Letter.)

1. Military Medical Training

Place: U.S. Naval Medical School, National Naval Medical Center, Bethesda, 14, Md.

Date: 11-24 March 1962

Description: First week will be devoted to Medical Aspects of Special Weapons and Radioactive Isotopes with particular reference to personnel casualties from atomic explosions. Second week will be devoted to professional topics of concern to military medicine, including discussions on Reserve Medical Programs of the Armed Forces.

Eligibility: Naval Reserve Medical Department officers. Quotas have been

authorized for the 1st, 3rd, 4th, 5th, 6th, 8th, and 9th Naval Districts and the Naval Air Reserve Training Command.

2. Seminar—Combined Seminar for Commanding Officers, or Their Representatives, of Naval Reserve Medical Specialist Units and of Hospital Corps Divisions and Commandant's Representatives at Medical Schools

Places and Dates: Fourth Naval District Headquarters, 10-12 July 1961; Twelfth Naval District Headquarters, 24-26 July 1961.

Description: To provide indoctrination and orientation in organization, administration, and operation of the Naval Reserve Program with emphasis on the medical components and the Navy Ensign Medical Program. Field trips to naval activities and other facilities will be conducted. A series of meetings will be held between the trainees and officers of the district staffs with a view toward an improved Medical Reserve Program through the exchange of ideas and recommendations.

Eligibility: Staff members of Naval Reserve Medical Specialist Units and Hospital Corps Divisions with priority being given to commanding officers and executive officers in that order, and Reserve officers serving

as Commandant's Representatives at medical schools. Quotas for the seminar at Headquarters, Fourth Naval District have been allocated to the 1st, 3rd, 4th, 5th, and 6th Naval Districts. Quotas for the seminar at Headquarters, Twelfth Naval District have been allocated to the 8th, 9th, 11th, 12th, and 13th Naval Districts.

### 3. On-the-Job Training - Officer

**Places and Dates:** Any suitable naval medical facility. Convening date to be arranged between commandant, trainee, and commanding officer of training activity.

**Description:** On-the-job training in Navy Medical Department operation and organization, with emphasis upon preparation of the trainee for potential mobilization duties.

**Eligibility:** Naval Reserve Medical Department officers, male and female, with previous ACDUTRA or active service. Quotas have been allocated to all naval districts (less 10, 14, 15 and 17).

### 4. Medical Department Orientation

**Places and Dates:** Any naval hospital in Continental United States. Convening date to be arranged between commandant, trainee, and commanding officer of hospital.

**Description:** This training is intended to indoctrinate and orient Naval Reserve Medical Department officers in the operation and function of the Medical Department of the Navy.

**Eligibility:** Naval Reserve Medical Department officers, male and female, who have not performed ACDUTRA or active duty. Quotas have been allocated to all naval districts (less 10, 14, 15, and 17).

### 5. On-the-Job Training in Submarine Medicine

**Place:** U.S. Naval Medical Research Laboratory, U.S. Naval Submarine Base, New London, Conn.

**Dates:** 7 August 1961  
6 November 1961  
5 February 1962  
7 May 1962

**Description:** On-the-job training presenting an up-to-date review of problems relating to submarine medicine, including recent developments in submarine medicine research.

**Eligibility:** Naval Reserve Medical Service Corps officers. Male only. Quotas for 7 August have been allocated to the 1st, 3rd, and 4th Naval Districts; quotas for 6 November have been allocated to the 1st, 3rd, 9th, and 11th Naval Districts; quotas for 5 February have been allocated to the 6th, 8th, 9th, and 12th Naval Districts; quotas for 7 May have been allocated to the 3rd, 4th, 5th, and 13th Naval Districts.

### 6. Tissue Bank Training Course

**Place:** U.S. Naval Medical School, National Naval Medical Center, Bethesda, Md.

**Dates:** 3 July 1961  
2 October 1961  
2 January 1962  
2 April 1962

**Description:** This course provides orientation in operation and administration of a tissue bank. It includes indoctrination in the methods of tissue procurement; storage and dispensing; tissue culture; tissue chemistry; processing excised tissue; and allied short and long-term culture and tissue chemistry fields. It also includes indoctrination in the medico-legal aspects of homotransplantation, the



procedure for obtaining permission for tissue donations, familiarization with the operation of the Tissue Bank Registry, and all other administrative practices associated with tissue banking.

Eligibility: Naval Reserve Medical Corps officers. Quotas have been allocated to all naval districts (less 10, 14, 15, and 17).

#### 7. On-the-Job Training - Field Medicine

Place: Marine Corps School, Quantico, Va.

Dates: Beginning any Monday morning July through September 1961 and June 1962.

Description: Indoctrination in amphibious warfare and field medical practices.

Eligibility: Naval Reserve Medical Corps officers. Male only. Quotas have been allocated to the 3rd, 4th, 5th, and 6th Naval Districts.

#### 8. Recognition and Treatment of Diving Casualties

Place: U. S. Naval School Deep Sea Divers, U. S. Naval Weapons Plant, Washington, D. C.

Date: 10 July 1961

Description: A two-week course offering didactic training in underwater physiology and in the recognition and treatment of casualties associated with any kind of diving. Instructions

include lectures and demonstrations of the equipment of the Deep Sea Divers School and Experimental Diving Unit. This course is given for active duty personnel enroute to stations where there is some diving activity; however, vacancies in the course may be filled by Reservists on active duty for training.

Eligibility: Naval Reserve Medical and Medical Service Corps male officer personnel. Quotas have been allocated to all Naval Districts (less 10, 14, 15, and 17).

#### 9. Military Nursing Symposium

Place: U. S. Naval Medical School, National Naval Medical Center, Bethesda, Md.

Date: 30 July - 12 August 1961

Description: This symposium is planned to acquaint the Navy Nurse Corps Reserve officers on inactive duty with the changes occurring within the Medical Department of the U. S. Navy. Lectures will include Administration of Nursing Service, Clinical Specialties, and Nursing Research. Information to be presented at this meeting will be current.

Eligibility: Naval Reserve Nurse Corps officers with preference to non-attenders of previous symposiums, but repeaters are eligible. Quotas have been allocated to all Naval Districts (less 10, 14, 15, and 17).

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Nutritional needs of older people are not different in kind from those of any age. They only differ in amount; proteins, fats, carbohydrates, vitamins, minerals, and salts all have their place in the diet.

—Ancel Keys



## OCCUPATIONAL MEDICINE

### Granulomatous Inflammation of Bursae

John E. Kirkpatrick MD, San Francisco, Calif. *Industr Med Surg* 30:89-92, March 1961.

Significant trauma to the elbow can cause dislocation or fracture of one or more components of this joint and result in considerable permanent disability. The healing process under effective management normally progresses steadily toward absorption of hemorrhage and edema, reunion of torn ligaments and fractures, and restoration of the smooth gliding tissues—synovia and bursae. Whatever permanent disability occurs does not result in development of "tennis elbow," "calcium deposit," bursitis, tenosynovitis, rheumatoid arthritis, or osteoarthritis. Most important of all, significant trauma does not produce any known inflammatory condition of the epicondyles of the humerus that can be proved by surgical exploration or microscopic examination of a biopsy of the epicondyle, or that can be visualized by roentgenograms.

After an injury such as this there may be some permanent restriction of the elbow motion due to ordinary scar tissue in the ligaments, but such a severe injury does not result in development of ganglion, chronic villous tenosynovitis, bursitis, calcific den-

sities in the bursae, stenosing tenosynovitis, the carpal tunnel syndrome, rheumatoid arthritis, or osteoarthritis.

Development of these diseases has often been reported as owing to a tear in a ligament of the elbow, bruises, bumps, occupational activity, repetitive motions, use of small hand tools, occupational microtrauma, overstretching of the muscles, exposure to cold, and many other extrinsic factors.

One complication occasionally seen after a Colles' fracture may occur—after about six weeks there may be a sudden rupture of the extensor pollicis longus tendon in its osseofibrous tunnel. The pathologic process involved in this complication is ischemic necrosis of the tendon, due to prolonged edema in its narrow compartment. Microscopic examination of the ruptured ends of the tendon in such cases does not reveal any evidence of granulomatous inflammation, but only simple tendon necrosis. This complication is not due to one of the chronic tendon or tendon sheath diseases caused by granulomatous inflammation.



However, spontaneous rupture of a tendon without significant trauma is always due to one of the granulomatous inflammations.

Let us weigh the dogma of trauma as a cause of several common entities against the facts of granulomatous inflammation involving the extremities.

A ganglion is often the first manifestation of degenerative disease of the ligaments of the wrist, hand, ankle, or foot. It usually makes a gradual appearance and pain is noticed if the area is struck; most frequently it is not noticed until some occurrence brings it to the attention of the patient. It cannot be produced by lacerating the carpal ligament or by surgical incision through the ligament. As a matter of fact, cure of it is effected by complete excision of the degenerated area of the carpal ligament together with the ganglion. Here, then, significant trauma (surgical) cures a condition presumed to be caused by trauma, a paradox. Recurrence of the ganglion in the same place is due to lack of complete excision of the degenerated portion of the carpal ligament causing the ganglion.

Taking into consideration the four extremities of each normal person, and the use and abuse they take in sports or work, the proportion of persons who have this disease is almost infinitesimal despite daily so-called microtrauma. Why perform surgical operation—a maximal trauma—if minimal trauma can cause this condition? Before surgical procedures were perfected for this condition it was common practice to pound the ganglion with a book to rupture it.

A collection of fluid in a tendon sheath is not a ganglion. Following significant trauma, either hemorrhage or a serious effusion may occur. This

condition runs a normal course of absorption which may be aided by aspiration with a needle; it does not recur at intervals and does not become chronic. Where an effusion in the tendon sheath does persist, or recur, then the diagnosis of traumatic origin becomes suspect. The pathologic reason for chronic tenosynovitis with effusion is that it is one of the granulomatous inflammations, currently classified as one of the rheumatic diseases. Crepitating, or villous tenosynovitis, with or without "rice bodies" (not due to tuberculosis or to any known animate agent) is due to one of the inanimate (chemical or physical) agents; the more common ones being cholesterol, uric acid, or rheumatoid factor, singly or in mixed lesions.

A granulomatous inflammation now thought to be produced by collagen (inanimate agent) is Dupuytren's contracture of the palmar or plantar fascia (also Peyroni's disease, and probably torticollis).

Dupuytren, a French surgeon, described this condition first in 1831. It affects less than 2% of the adult population, and occurs chiefly in men. It is seldom seen in Negroes or in Indians. Most persons who have it are unaware of the early manifestations of this disease unless they experience an acute inflammatory attack in one or more nodules of the fascia, accompanied by tenderness and redness of the overlying skin.

The acute inflammatory phase of this disease is characterized by pain, redness of the skin, and localized swelling over the area of the involved palmar fascia. It may be the first attack before fibrosis of the fascia has developed. It can occur also as

an acute exacerbation of the disease in advanced fibrosis of the palmar fascia which has already produced contractures.

Recent microscopic sections of palmar fascia which I have obtained surgically during the acute inflammatory phase of Dupuytren's disease reveal acute vasculitis.

Although a therapeutic dose of x-ray can cause the inflammatory reaction to disappear promptly, its use on the hand is frowned upon except in individuals whose life expectancy is not more than 20 or 30 years because of the latent carcinogenic effect of skin radiation. The effect of x-ray therapy on the acute stage of vasculitis does not retard the progression of fibrosis and contracture of the diseased palmar fascia.

Following injury of a hand or wrist requiring a few weeks of bandaging or being in a plaster cast, the patient may suddenly discover the condition and honestly believe he never had it before because he has been unable to appreciate that it had begun insidiously several years previously. In such cases it usually can be found—in one stage or another—in the uninjured hand. It is most often bilateral and may be more advanced in one hand than the other. The major hand is not always involved to a greater extent than the opposite.

Gout is a systemic disease due to an inborn inability to excrete uric acid normally. Approximately a half million Americans are afflicted with some form of gout. A common misconception of manifestations of gout is that it only occurs in the great toe, or that tophi can only be found in the ears. This granulomatous inflammation caused by uric acid (inanimate agent)

can manifest itself in any joint and in any bursa or synovial space (as well as many other parts and organs of the body including the heart and the entire vascular system). In those afflicted with tophaceous gout, uric acid deposits are found in one or multiple locations, commonly subchondral areas adjacent to the collateral ligaments of joints, and in bursal and synovial spaces. Such a deposit in one of these areas may form a weeping sinus through the skin, which is seen most frequently in the digits but sometimes in the skin over the olecranon bursa. Gouty peri-arthritis may be confused with other forms of arthritis.

If an acute inflammatory episode occurs which responds to adequate dosage of colchicine, it is certainly very likely acute gout; if the patient does not remember—or does not wish to remember—any previous attacks, it may be the first attack. Occasionally gout is associated with the granulomatous inflammation of rheumatoid arthritis, the cause of which is unknown, but recently has been associated with the rheumatoid factor (inanimate agent).

Direct significant trauma to a subcutaneous bursa usually ruptures the bursa lining and the sac fills with blood. This resolves with or without aspiration in a short period of time and does not recur or become chronic. It can occur again after another trauma, but a new hemorrhage cannot occur spontaneously without rupture of a blood vessel. An acutely swollen bursa with warm red skin overlying it is most frequently caused by the granulomatous inflammation of uric acid, cholesterol or both, and is not due to "think back" trauma, or



"must have bumped it" trauma. In bursitis of this type microscopic studies of the lining of the bursa prove the diagnosis. Specific therapeutic treatment causes the inflammatory reaction to subside rapidly. Opacities seen in x-ray films of these areas often are reversible by systemic medication.

Adventitious bursae can be caused by specific repeated occupational trauma (as in hod-carrying, hardwood floor laying) but these bursae do not develop granulomatous inflammations.

This question has always been helpful when making a diagnosis: What would you, as a physician, expect would happen following a certain describable trauma; and, drawing on your experience in many similar cases, what has been the effect of this type of trauma on the part so injured?

It is doubtful if any physician would subscribe to a statement that a blow that fractured ribs over the heart could cause rheumatic endocarditis. However, there are many who will subscribe to a statement that a severe blow or even a single minor one, or constant minor trauma, in the region of a joint can cause the development of some chronic condition, whatever it may be—bursitis, epicondylitis, tenosynovitis, calcific tendonitis, gouty periarthrititis, or rheumatoid or degenerative arthritis.

The accepted medical dictionary definition of "tennis elbow" is an acute or chronic synovitis of the radial humeral bursa.

Barritt said that a physician should render a medical opinion based on reasonable medical probabilities. (Be it noted he did not say possibilities).

It takes more mental energy to think about the role of the reticuloendothelial cells and their proliferative re-

action which displaces normal cells in tissues in granulomatous inflammatory disease than to accept common offhand epithets such as "wear-and-tear" or "microtrauma" in the etiologic description of these conditions. Forbus suggested that a further understanding of the granulomatous inflammatory reaction may lead to a solution of the cause of genuine neoplasia.

There are a number of other conditions which have been blamed on trauma, among them xanthoma of tendons, which is caused by a hypercholesteremic familial metabolic disorder; spontaneous rupture of tendons in their osseofibrous tunnels from rheumatoid arthritis; peripheral median nerve neuritis due to rheumatoid, or other types of tenosynovitis of the flexor tendons in the carpal tunnel; trigger finger; DeQuervain's disease; nonosteogenic fibroma of bone; and Heberden's nodes.

Granulomatous inflammation can be produced by many agents, but not by trauma—by animate agents such as bacteria and coccidia; or by silica, fats and oils, collagen, cholesterol, uric acid, rheumatoid factor, Bence-Jones protein; or by unknown agents, as in Hodgkin's disease, rheumatic fever, mycosis fungoides, and sarcoid of Boeck.

Some of the more common types of disease affecting the extremities during years of employment are fibrositis, myositis, bursitis and so-called epicondylitis, ("tennis elbow"), which affect only a small portion of the 11 million Americans afflicted with arthritis and related rheumatic diseases. Some of these conditions run a course independent of treatment; some of the effects of the disease are



reversible without treatment. Most of them have a tendency to recur at more or less frequent intervals, or to be chronic. None of the rheumatic diseases or granulomatous inflammations presented here as examples have ever been created by slight or severe mechanical trauma either in experimental animals or in human beings.

One recent article on the "carpal tunnel syndrome" stated that division of the carpal ligament in 16 cases afforded "immediate and lasting relief" from the symptoms. It is amazing to read further and find this statement: "In every instance the (flexor) tendons were covered by an abnormally thick sheath of material, the nature of which is not known."

This condition, "the nature of which is not known," is too often generally ascribed to trauma, repetitive motion, or some similar unscientific etiology. The condition is most commonly due to one of the rheumatic diseases producing a tenosynovitis; it is caused frequently by the rheumatoid factor and less commonly by gout or cholesterol granulomatous inflammation.

These diseases are associated with dysfunction of the reticuloendothelial system, and trauma does not produce a persistent stimulative effect on the cells of the reticuloendothelial system. The course of the granulomatous inflammations is toward chronicity and recurrence. These lesions may be reversible, may end in scarring, or may cause total destruction of the individual.

The problem lies not so much in a lack of pathologic reasoning and understanding as in specious socioeconomic reasoning—almost as if when a patient says something is so, his physician must say it is so in order to keep out of an argument that may affect his economic status and thus avoid conflict with the patient, a labor union, or the patient's employer. It is granted that the impact of such reasoning was greater during the earlier part of the past 50 years of the age of industry, for in those days there was limited insurance coverage, particularly for off-the-job occurrences. Now, with most industrial employees covered by an umbrella of insurance both on and off duty, fewer and fewer young physicians will be exposed to this unsound concept, and it is now more likely that the physician will be remunerated for services that once would not have been paid for if the condition were certified as not being industrially connected from the standpoint of scientific fact and probability.

When a patient who attributes a disease of the extremities to on-the-job trauma is found, upon physical examination, to have one of the granulomatous diseases dealt with herein, rather than a "compensable" injury, the physician can do the patient a worthwhile service by making an accurate diagnosis and informing him of the real nature of his illness.

As Thannhauser said, "Every patient is a new experience and a challenge to the keenness of our senses."

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The brain is a wonderful organ. It starts working the moment you get up in the morning, and does not stop until you get into the office.

— Frost



### Control Measures to Reduce Occupational Heat Stress

Ways of manipulating environmental factors to reduce heat stress to workers in hot jobs and some methods used in training and scheduling personnel to avoid undue strain were presented in a paper by B. A. Hertig and H. S. Belding Ph.D., of the University of Pittsburgh (Pittsburgh 13, Pa.) at the Symposium on Temperature and Its Measurement and Control in Science and Industry, held in Columbus, Ohio, 27-31 March 1961. The meeting was sponsored by the American Institute of Physics, the Instrument Society of America, and the National Bureau of Standards. Points listed by the authors are summarized:

1. By interposing reflective or absorptive shields between source and worker, dramatic reductions in heat stress caused by radiant energy from surfaces of hot bodies can be achieved. Alternatively, radiating surfaces can be treated to reduce emissivity.
2. Adequate rate of change of air surrounding the hot process can prevent excessive rise in air temperatures. Air exhaust units, however, will not be adequate unless there is also provision for sufficient make-up air.
3. Insulation affords a means for reducing the convective heat loss from moderately hot bodies, but because of thickness required, it is impractical for high-temperature sources.
4. Mechanical refrigeration is economically feasible for certain work spaces such as crane cabs and panel control areas. Air-conditioned rest areas favor a rapid return of body temperature to base line. Conditioned air circulated through special clothing offers the most efficient use of refrigerated air.
5. Increasing air speed increases the evaporative capacity of the environment which is important in warm-humid environments such as mines, laundries, and the tropics. Doubling air speed results in a 50% increase in evaporative capacity, but at 4 times power requirements.
6. Breaking in new employees gradually until they become acclimatized to the heat results in dramatic decreases in strain of exposures.
7. Where thermal balance with the environment is not possible, work schedules must be arranged to provide workers with periodic respites away from the heat so that stored body heat may be dissipated and excessive rise in body temperature prevented. It has been demonstrated that short work-rest cycles permit more work in the heat per shift.
8. Ordinary khaki work clothing, though "black" to long wave radiant energy, reduces the effective radiant heat load by some 30%. Clothing also provides insulation against convective gain when air temperature exceeds skin temperature.
9. For protection against solar radiation for persons engaged in out-of-doors occupations, light-colored clothing will reflect energy in the visible spectrum; but, as with khaki, it is essentially "black" to long-wave emanations. (Industr Hyg News Rpt, May 1961)

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Chlorinated Plastic Paint May Be  
Unexpected Fire Hazard

Strippable coatings, consisting of chlorinated plastic materials such as vinyl chloride acetate, are extensively used on walls and ceilings of laboratories where a possibility of contaminating spills exists. Should the surfaces become contaminated the outer layer of the paint can be peeled or stripped off, thus removing the contamination.

In a recent fire the strippable coating burned off all the walls from about 3 feet above the floor, up to and across the ceiling in a manner typical of surface-coating fires. One observer stated that the coating "hung in strips like fly paper." The gypsum wallboard did not burn through. However, dense smoke and extremely strong hydrogen chloride "vapors" were reported.

According to literature supplied by the manufacturer of this paint, it affords "excellent fire protection, as it will not support combustion." In a recent discussion with the manufacturer's representative, he stated that at high temperatures the paint would evolve CO<sub>2</sub> which would act as a fire extinguisher. No indication was given, however, that additional products of combustion might be toxic, combustible, and corrosive.

The products of combustion of chlorinated plastics have been studied and reported in the literature. E. H. Coleman and C. H. Thomas (Journal Applied Chemistry 4:379-383, July 1954, report that at the estimated temperature of this fire (1200 F) various chlorinated plastics (based on 1.05 grams material per 5-1/2 liters air) generate concentrations of hydrochloric acid ranging from 7000 to

8000 parts per million by volume (ppm), carbon monoxide ranging from 6000 to 22,000 ppm, and carbon dioxide from 15,000 to 28,000 ppm. Of course, temperatures and fuel-air ratios of such fires vary and therefore result in different concentrations; but an indication of the decomposition products is evident from this report.

Subsequent to the fire, radiant-panel, flame-spread tests (Federal Standard No. 00136, Com-NBS, July 31, 1959, or ASTM E-162) were made using 6 by 18-inch panels of gypsum wallboard and hardboard coated with the paints to be tested. In this test the panels are mounted slightly inclined from the vertical and heated with a radiant panel at 1238 F, while evolved gases, if any, are ignited from above. This test is considered analogous to walls and ceilings being exposed to the heat of a fire occurring indoors.

Results of this series of tests show that those panels coated with chlorinated plastic paint flash vigorously over 1/3 to 1/2 of the panel and drip flaming material. The coating increased the flame-spread rating of uncoated gypsum board from an average of 9 to an average of 50. (Transite = 0, red oak = 80 - 160).

Currently, commercially available strippable coatings are being evaluated as noted above and, in addition, toxicologic evaluations are being made of both the material and any possible decomposition products. Insufficient attention has been paid in the past to this hazard aspect of strippable coatings. (Hazards Control Information Exchange Bulletin, March 1961).



Occupational Medicine Briefs**The Sunlight Factor in Aging and Skin Cancer**

Ultraviolet radiation of 3200 Angstrom units and shorter in sunlight is believed to be the cause of skin cancer and aging of the skin in man. This is based on experimental production of malignant tumors in rats and mice by these wavelengths, as well as on 4 clinical observations: (1) predilectional localization of skin cancer to exposed sites; (2) greatest incidence in geographic areas that receive the greatest insolation; (3) greatest incidence among outdoor and rural workers; and (4) higher frequency in light-skinned than in dark-skinned persons. The primary site of action of the ultraviolet radiation may be the subpapillary vascular plexuses of the dermis, which, after injury, disturbs the nutrition of the overlying epidermis and causes aging of the skin, keratoses, and eventually squamous and basal-cell epitheliomas in susceptible persons. Light-skinned persons who tan poorly should avoid the sun or take protective measures. (J. B. Howell, Arch Dermatol 82:865, December 1960; abstracted in Industr Hyg Dig, April 1961)

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**Chrome Sensitivity in Cement and Oil Dermatitis**

The writer highlights several perplexing aspects of the part played by chrome sensitivity in cement and oil

dermatitis. In a series of 66 patients with dermatitis thought to be caused by cement, 95.4% reacted positively to patch tests with 0.1% potassium dichromate. In a series of 102 patients with eczema from other causes, he reports no positive reactions to the same test! The weakest solution of potassium dichromate to show consistent results was 0.1%, although 13 of 15 patients reacted to a 0.05% solution and 5 of 15 tested reacted minimally to a 0.01% solution. Spectrographic analysis of offending cements showed presence of chromium in all samples tested. Colorimetric technics were utilized to measure the amount of chromium in 18 samples of local cement; the soluble hexavalent chromium content was of the order of 3 to 4 mg/g of cement. Alkaline milieu, friction, evaporation, and other factors are postulated as tending to increase the amount of chromium making contact with the skin. In a series of 134 patients with oil dermatitis, 6% showed positive patch test reaction with 0.1% potassium dichromate. Six of the 8 positive reactors were donkey-greasers on ships when they developed the dermatitis. Since the author was unable to demonstrate any chromium in oils, "even those oils taken from the engines of ships," he concludes that anti-rust agents containing chromium were the possible sources of chromium. (F. E. Anderson, Brit J Dermatol 120: 108, March 1960; abstracted in Industr Hyg Dig, December 1960)

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### Radiologic Diagnosis in Asbestosis

The authors studied lung function in 40 patients at the Royal Free Hospital, London, who had been exposed to asbestos dust in their work for varying periods of time. Of these, asbestosis had been certified by the pneumoconiosis medical boards in 21. Ten patients with a prolonged exposure did not have definite roentgenographic signs of asbestosis; in 9 patients the diagnosis of asbestosis was doubtful. Changes of pulmonary function observed in patients with asbestosis were those common to patients with other interstitial fibroses. They consisted of a lowered diffusing capacity together with a reduced inspiratory capacity, and hyperventilation on exertion

(often accompanied by arterial desaturation) with no evidence of air-flow obstruction except in patients in whom asbestosis was complicated by asthma or emphysema. Many patients also showed inequality of ventilation and ventilation-perfusion ratio. These functional changes were related to an independent grading of physical signs and roentgenographic appearances. Changes in lung function, specific for interstitial fibrosis, often precede definite changes in the chest roentgenogram, so that finding them in patients with a history of exposure to asbestos is suggestive evidence of asbestosis whatever the roentgenographic appearances may be. (R. Williams and P. Hugh-Jones, Thorax, June, 1960; abstracted in Industr Hyg Dig, March 1961)

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